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Infarct Location and Sleep Apnea: Evaluating the Potential Association in Acute Ischemic Stroke

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Abstract

Background—The literature about the relationship between obstructive sleep apnea (OSA) and stroke location is conflicting with some studies finding an association and others demonstrating no relationship. Among acute ischemic stroke patients, we sought to examine the relationship

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between stroke location and the prevalence of OSA; OSA severity based on apnea-hypopnea index (AHI), arousal frequency, and measure of hypoxia; and number of central and obstructive respiratory events.

Methods—Data were obtained from patients who participated in a randomized controlled trial (NCT01446913) that evaluated the effectiveness of a strategy of diagnosing and treating OSA among patients with acute ischemic stroke and transient ischemic attack. Stroke location was classified by brain imaging reports into subdivisions of lobes, subcortical areas, brainstem, cerebellum, and vascular territory. The association between acute stroke location and polysomnographic findings was evaluated using logistic regression for OSA presence and negative binomial regression for AHI.

Results—Among 73 patients with complete polysomnography and stroke location data, 58 (79%) had OSA. In unadjusted models, no stroke location variable was associated with the prevalence or severity of OSA. Similarly, in multivariable modeling, groupings of stroke location were also not associated with OSA presence.

Conclusions—These results indicate that OSA is present in the majority of stroke patients and imply that stroke location cannot be used to identify a group with higher risk of OSA. The results also suggest that OSA likely predated the stroke. Given this high overall prevalence, strong consideration should be given to obtaining polysomnography for all ischemic stroke patients.

Keywords

stroke; infarct; CT; MRI; sleep apnea

1. Introduction

Obstructive sleep apnea (OSA) has been documented to be present in 73–93%[1, 2] of ischemic stroke patients, compared with a community prevalence of 21%.[3] Patients with acute ischemic stroke and OSA have worse outcomes than those without OSA, including increased risk of early neurological worsening, mortality, decreased functional recovery, nonfatal cardiovascular events, longer hospital stays, delirium, and depressed mood.[4–10] Because early neurologic worsening is likely a reflection of injury in the ischemic penumbra, some authors have postulated that early identification and treatment of OSA may prevent clinical deterioration.[4, 7] Use of continuous positive airway pressure in acute ischemic stroke patients with OSA has been shown to improve certain post-stroke outcomes.[8, 11–14]

In contrast to the robust and consistent evidence about OSA prevalence post-stroke, the literature about the relationship between OSA and stroke location is conflicting with some studies finding an association[2, 5, 9, 15–17] and others demonstrating no relationship.[4, 10, 18, 19] Most studies included small sample sizes. Some reported non-statistically significant associations between certain locations and OSA presence, and other studies included very broad location categories (e.g., comparing large vascular territories or infratentorial versus supratentorial locations). The objective of the current study was to evaluate the potential relationships between stroke location and a variety of polysomnographic characteristics. Specifically, among acute ischemic stroke patients, we sought to examine the relationship

between stroke location and the prevalence of OSA; OSA severity based on AHI, arousal frequency, and measure of hypoxia; and number of central and obstructive respiratory events.

2. Methods

2.1. Sample

Data were obtained from patients who participated in the intervention group of a randomized controlled trial (NCT01446913) that evaluated the effectiveness of a strategy of diagnosing and treating OSA among patients with acute ischemic stroke or transient ischemic attack (TIA). As part of the trial, intervention patients received polysomnography. The study was conducted at 6 participating hospitals in Connecticut and Indiana. Screening occurred within 1 week of stroke symptom onset. Patients included in the study were those subjects 18 years of age or older with an admitting or working diagnosis of TIA or stroke. Exclusion criteria included patients with known sleep apnea or other sleep disorder diagnosis, patients going to hospice or receiving comfort measures only, inability or unwillingness to use a face mask, use of mechanical ventilation, non-English speaker/comprehension, inability of the subject to give informed consent to participate in the study, and suicidal ideation. This sub-study focused on patients with brain imaging evidence of acute ischemia who had polysomnographic data. This study had institutional review board approval for the use of human subjects at all participating sites. Informed written patient consent to perform this study was received from all study participants.

2.2. Data Collection

All medical history variables were obtained either from clinician notes from the emergency department (ED)/admission period or from patient report. Patients were assessed using a standard protocol that included a structured patient interview, an in-depth medical record review, and a clinical examination. Interview questions and medical record review included history of comorbidities (hypertension, hypercholesterolemia, diabetes mellitus, peripheral vascular disease, asthma/chronic obstructive pulmonary disease, coronary artery disease, atrial fibrillation, congestive heart failure), tobacco or alcohol use, prior stroke or TIA, and time of stroke onset. The clinical examination included manual blood pressures, heart rate, neck and waist measurements, body mass index (BMI), and assessment of stroke severity using the National Institutes of Health Stroke Scale (NIHSS).

2.3. Stroke Location

For patients to be included in the trial, brain imaging by computed tomography (CT) or magnetic resonance imaging (MRI) needed to be completed within 48 hours of presentation to the ED or hospital. Brain imaging reports were recorded in the study database; these were reviewed for the purpose of identifying stroke location by a neurologist (S.M.S.) who was blinded to the clinical and sleep data at the time of the brain imaging review. In unadjusted modeling, acute ischemic stroke location was classified on the basis of brain imaging (MRI or CT) reports as follows: lobar (frontal, parietal, temporal, insular, occipital), subcortical (thalamus, basal ganglia, internal capsule, corona radiata, corpus callosum), brainstem (midbrain, pons, medulla), and/or cerebellum/cerebellar peduncle. Some reports only listed

the vascular territory [middle cerebral artery (MCA) territory, anterior cerebral artery (ACA) territory, and/or posterior cerebral artery (PCA) territory] and, therefore, could not be classified into a more specific location. Some reports did not list the vascular territory. Lobar locations included cortical and/or subcortical infarcts. Additionally, some patients had strokes involving multiple lobes; therefore, each lobe was documented as being involved (e.g., a patient with an MCA stroke involving the frontal, temporal, and parietal lobes was classified in each of these 4 territories – MCA, frontal, temporal, and parietal). Groups of stroke location were defined as listed in Table e-1 into the following categories: posterior circulation, anterior circulation, subcortical/lacunar, supratentorial, infratentorial, and potential motor pathways. Potential motor pathways were evaluated in an attempt to distinguish pathways that could contribute to bulbar/upper airway weakness and were defined as areas in which motor tracts could potentially be involved (e.g., frontal lobe, thalamus, internal capsule, and brainstem locations). The stroke locations used in this study were chosen to provide more specific locations compared to many studies that used large groupings of location, such as large vascular territories, cortical/subcortical/or brainstem, and supratentorial or infratentorial locations.[2, 4, 5, 8, 10, 12–14, 16, 18–20] Given that brain imaging reports do not consistently provide a measurement of infarct size, no specific assessment of infarct size could be included in these analyses except as inferred by the number of lobes involved.

2.4. Sleep Evaluation

Sleep workup included a comprehensive interview and questionnaires that inquired about sleep habits and disturbances. The Epworth Sleepiness Scale was used to evaluate daytime sleepiness preceding the stroke and was considered abnormal if the score was > 10.[21] Patients underwent an unattended sleep study to diagnose sleep apnea using the Safiro™ sleep monitor(Compumedics, Victoria Australia). This monitor is a type 2, full 18 channel unattended sleep monitor including 4 channels of electroencephalography, electrooculography, electromyography, snore channel, oral thermistor, nasal pressure, chest and abdominal effort, leg movements, position, electrocardiogram, and oxygen saturation. All studies were scored at a central reading location at Harvard University School of Medicine, and the definitions of respiratory events conformed to those of the American Academy of Sleep Medicine.[22] An apnea was defined as a complete cessation of airflow for ≥ 10 seconds. A hypopnea was defined as a decrease in nasal pressure of ≥ 30% from baseline associated with ≥ 3% oxygen desaturation or an arousal. Apneas and hypopneas were scored as either obstructive or central to indicate the presence or absence of respiratory effort respectively.[22] According to the recommendations from the American Academy of Sleep Medicine Task Force and the Internal Classification of Sleep Disorders, 3rd edition, the AHI was defined as the average number of apneas and hypopneas per hour of sleep. OSA was defined as having an AHI of ≥ 5 events per hour with predominantly obstructive respiratory events in a patient diagnosed with stroke. Central sleep apnea (CSA) was defined if both of the following were present: 1) ≥ 5 central apneas and/or central hypopneas per hour of sleep and 2) the number of central respiratory events was > 50% of the total number of apneas and hypopneas.[23, 24]

2.5. Statistical Analysis

Descriptive statistics (e.g., mean with standard deviations, frequencies and proportions) were used to describe the baseline characteristics and polysomnographic outcomes. To compare differences in polysomnographic outcomes by either baseline characteristics or stroke location, Chi-square tests or Fisher's exact tests were used for binary variables (e.g., OSA presence/absence), and Student's t-tests or Wilcoxon rank sum tests for continuous variables (AHI in events/hour). Two-sided *p*-values were used without corrections for multiple comparisons. We conducted multivariable modeling to assess the relationship between stroke location and OSA presence and AHI. Specifically, we used logistic regression to model OSA presence, adjusting for baseline characteristics that were associated with OSA. No imputations were made for missing data. All analyses were conducted using SAS version 9.2 (Cary, North Carolina). Given that this was an exploratory analysis of an existing dataset, we did not conduct formal sample size analyses. However, in the construction of all multivariable models, we maintained a 10:1 event per variable ratio.[25]

3. Results

Overall, 135 patients were diagnosed with acute stroke. One hundred sixteen patients had evidence of acute ischemia on brain imaging, of whom 73 had polysomnographic data (Figure 1). The baseline characteristics of those with polysomnography did not differ significantly from those without polysomnography. Polysomnography was performed at a median of 37 days after stroke onset. Table 1 displays the baseline characteristics of the 73 patients in the whole sample and the 58 patients (79%) with OSA and the 15 patients without OSA. The overall (mean±standard deviation) AHI was 12.2±13.9 per hour of sleep, ratio of central to obstructive respiratory events (i.e., hypopneas and apneas) 0.4±1.4 per hour of sleep, obstructive apnea index 4.6±10.4 per hour of sleep, central apnea index 1.1±4.2 per hour of sleep, arousal index 20.9±11.7 per hour of sleep, percent time with oxygen saturation < 90% 3.9±8.6 minutes, and average oxygen saturation 94.2±1.9 percent.

The 73 patients included 16 women and 57 men with a mean age of 59.5±11.6 years. The following baseline characteristics differed significantly between patients with OSA and those without OSA: neck circumference (16.3±1.9 inches with OSA, 14.7±1.1 without OSA, p=0.002), large neck size (defined as >16″ in women and >17″ in men) (38% with OSA, 0% without OSA, p=0.003), presence of facial weakness (25% with OSA, 60% without OSA, p=0.01), and presence of dysarthria (14% with OSA, 47% without OSA, p=0.01). There were no significant differences between patients with or without OSA in terms of the numerous other baseline characteristics including stroke severity as assessed by the NIHSS (Table 1).

In unadjusted models, no stroke location variable was associated with the prevalence of OSA (Table 2). Seventy out of the 73 patients had a specific stroke location documented; only 3 patients had only a vascular territory documented on the radiology report. No patients were classified as having CSA. Stroke size, as indicated by number of lobes involved, was not associated with OSA prevalence. Sleep apnea severity as measured by the AHI was also not associated with stroke location (Table 3). Similarly, neither the arousal frequency, percent time with oxygen saturation < 90%, nor the average oxygen saturation were

associated with stroke location (Table 3). In multivariable analysis, adjusting for neck circumference and BMI, groups of locations (i.e., anterior circulation, posterior circulation, lacunar, supratentorial, infratentorial, and areas containing motor pathways) were not associated with OSA prevalence or its severity (Table 4).

4. Discussion

The prevalence of OSA in our study was 79%. These results confirm that OSA is present in the majority of acute ischemic stroke patients. The results of this study suggest that a single stroke location or groupings of stroke locations cannot be used to identify a patient population with higher risk of OSA. Larger stroke size as indicated by the number of lobes involved and stroke severity based on NIHSS also did not correlate with a higher prevalence of OSA. Case reports and a small study of 10 patients suggested that the presence of bulbar weakness after a stroke leads to OSA.[26–28] Indeed, hypothetically, a stroke that impairs the pharyngeal muscle tone may predispose to upper airway obstruction and OSA. However, our data suggest that the presence of dysarthria and/or facial weakness was actually more common in those without OSA and, therefore, cannot be used to identify those with a higher risk of OSA. Given the relationship between OSA and both hypertension and insulin resistance, one might hypothesize that patients with OSA might be at increased risk of lacunar infarcts. Consistent with this hypothesis, Harbison et al. demonstrated significantly worse OSA in patients with lacunar infarcts than in patients with anterior circulation cortical strokes.[16] Other studies have also reported an association between sleep apnea severity and lacunar infarction.[2, 15] Our data did not support an association between subcortical/lacunar stroke location and either OSA prevalence or AHI.

Few baseline characteristics were associated with the presence of OSA in this sample of acute stroke patients. No medical comorbidity reached statistical significance, suggesting that comorbidities cannot be used in acute stroke patients to identify those who are at a higher risk of OSA. These findings are in concordance with other studies that have also demonstrated that baseline characteristics that are typically associated with OSA in the general population (e.g., BMI, age, smoking, alcohol consumption, daytime sleepiness, and hypertension) are not predictive of OSA in the acute stroke population.[7, 29, 30] These results suggest that patients with OSA and stroke may represent a different OSA phenotype and imply that typical screening questionnaires for OSA are less useful among patients with stroke, necessitating some form of polysomnographic testing for OSA case identification.

No patients were classified as having central sleep apnea among the 73 patients in this study. This finding is contrary to the common belief that strokes often cause central sleep apneas and Cheyne-Stokes respirations, especially strokes in the brainstem area.[26, 31, 32] In this study, no central apneas were present in the patients with brainstem strokes. Many other studies have reported that whereas obstructive and mixed sleep apnea is common post-stroke, central sleep apnea is uncommon post-stroke.[4, 19, 33]

Overall these findings are similar to prior studies, which failed to find an association between stroke location and OSA prevalence.[4, 9, 20, 34] One probable explanation is that the OSA was a preexisting condition (present yet undiagnosed prior to the stroke). Several large

cohort studies have demonstrated that OSA is an independent risk factor for stroke,[35, 36] providing support for the idea that the OSA predated the stroke in the vast majority of patients and therefore stroke characteristics (e.g., location and severity) are unlikely to be strongly associated with OSA characteristics.

Several limitations of this study require attention. The patient population included 73 patients, and only 8 strokes were located in the brainstem. A recent study by Brown et al. demonstrated brainstem involvement of stroke was associated with a higher prevalence of OSA and a higher AHI compared to strokes without brainstem involvement.[17] However, the small number of brainstem strokes in the current study is similar to that documented in other studies and is representative of the percentage of ischemic strokes that occur in the brainstem.[2, 16, 18, 19] Additionally, the assessment of stroke location was limited to that which was documented in the brain imaging reports (in other words, the radiographic studies were not available for central review) and volumetric assessment of infarct size was not available. Only 15 of the 73 patients did not have OSA, creating a low statistical power to determine significant differences between OSA and non-OSA groups. Another potential limitation was the use of the unattended polysomnography. However, the device used in this study provided all of the information that is typically included in formal in-laboratory polysomnography with the exception of videography. The polysomnograms were all read by a central laboratory using standard definitions. Moreover, the study population may not be representative of the general stroke population given that these patients were enrolled in an OSA treatment trial.

Although the association between OSA and stroke has been repeatedly demonstrated, many acute stroke patients do not routinely receive polysomnography despite recent stroke guidelines recommending consideration of polysomnography in all patients with an ischemic stroke.[37] The early identification and treatment of OSA has been shown to improve post-stroke outcomes.[8, 11–14] The results of this study confirm that OSA is present in the majority of stroke patients and suggest that stroke location and characteristics, including size, cannot be used to identify a group with a higher risk of OSA. Moreover, commonly used risk factors for OSA in the general population do not appear to apply to patients with stroke. Given the overall high presence of OSA, lack of clearly identifiable predictive factors, and evidence that treating OSA appears to improve certain post-stroke outcomes, strong consideration should be given to providing polysomnography for all patients with acute ischemic strokes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Abbreviations (in text)

ACA	anterior cerebral artery
AHI	apnea-hypopnea index
BMI	body mass index
CSA	central sleep apnea
CT	computed tomography
ED	emergency department
MCA	middle cerebral artery
MRI	magnetic resonance imaging
NIHSS	National Institutes of Health Stroke Scale
OSA	obstructive sleep apnea
PCA	posterior cerebral artery
TIA	transient ischemic attack

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HIGHLIGHTS

- The relationship between infarction location and sleep apnea was examined.
- Obstructive sleep apnea (OSA) is present in the majority of stroke patients.
- Infarct location cannot reliably be used to identify a higher risk of OSA.
- There are no clearly identifiable OSA predictive factors in acute stroke patients.
- Polysomnography should be considered in all acute stroke patients.

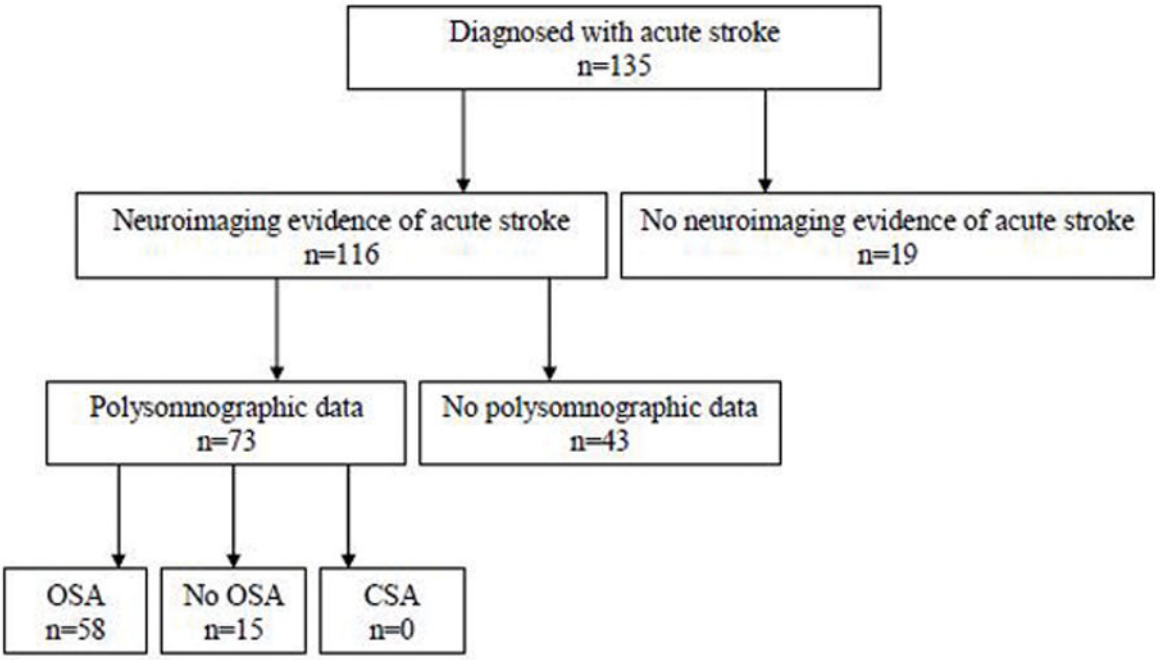

Fig. 1.

Diagram of patient recruitment and retention. CSA: central sleep apnea; OSA: obstructive sleep apnea.

Table 1

Baseline characteristics of the patients.

Characteristic	Overall (n=73)	OSA[a] (n=58)	No OSA (n=15)	*p*-value
AHI	19.8±18.9	24.5±18.7	2.2±1.6	<0.0001[b]
Age, years	59.5±11.6	60.4±11.4	56.2±12.3	0.22
Female sex	16 (21.9)	12 (20.7)	4 (26.7)	0.73
Body mass index, kg/m^2	30.3±7.5	30.1±7.2	30.9±7.2	0.73
Large neck size[c]	22 (30.1)	22 (37.9)	0	0.003[b]
Waist, inches	42.1±5.9	42.7±6.0	40.0±5.3	0.13
Hypertension	41 (56.2)	34 (58.6)	7 (46.7)	0.16
Hyperlipidemia	38 (52.1)	31 (55.2)	6 (40.0)	0.39
Diabetes mellitus	18 (24.7)	14 (24.1)	4 (26.7)	0.99
Peripheral vascular disease	7 (9.6)	7 (12.1)	0	0.33
Asthma/COPD	8 (11.0)	7 (12.1)	1 (6.7)	0.99
Coronary artery disease	15 (20.6)	14 (24.1)	1 (6.7)	0.17
Atrial fibrillation	5 (6.9)	5 (8.62)	0	0.58
Congestive heart failure	5 (6.9)	4 (6.9)	1 (7.1)	0.99
History of tobacco use	49 (67.1)	39 (67.24)	10 (66.7)	0.99
Current alcohol use	34 (46.6)	28 (48.3)	6 (40.0)	0.77
History of prior stroke/TIA	15 (20.6)	12 (20.7)	3 (20.0)	0.99
ESS score > 10	68 (93.2)	56 (96.6)	12 (80.0)	0.06
High risk for OSA on Berlin Questionnaire	47 (69.1)	36 (65.6)	11 (84.6)	0.32
PHQ-9	4.4±4.0	3.9±3.0	6.1±5.1	0.07
Initial NIHSS	2.3±2.3	2.1±2.3	2.8±2.3	0.31
Modified Rankin Scale	1.9±1.3	1.9±1.3	1.9±1.3	0.98
tPA during hospitalization	9 (12.3)	6 (10.3)	3 (20.0)	0.38
Low Ejection fraction[d]	28 (38.4)	20 (34.5)	8 (53.3)	0.24
Presence of PFO[e]	7 (9.6)	6 (10.3)	1 (6.7)	0.99
Initial SBP, mmHg	133.8±19.6	134.4±20.8	131.2±14.5	0.58
Initial DBP, mmHg	80.9±8.6	80.8±8.9	81.4±7.4	0.81
Presence of wake-up stroke	2 (2.7)	2 (2.7)	0	0.99
Facial weakness on NIHSS	23 (31.9)	14 (24.6)	9 (60.0)	0.01[b]
Dysarthria on NIHSS	15 (20.8)	8 (14.4)	7 (46.7)	0.01[b]
Presence of SVID	40 (54.8)	34 (58.6)	6 (40.0)	0.20

Shown are mean±SD or n (%). AHI: apnea-hypopnea index; COPD: chronic obstructive pulmonary disease; DBP: diastolic blood pressure; ESS: Epworth Sleepiness Scale; NIHSS: National Institutes of Health Stroke Scale; OSA: obstructive sleep apnea; PFO: patent foramen ovale; PHQ-9: Patient Health Questionnaire-9; SBP: systolic blood pressure; SVID: small vessel ischemic disease; TIA: transient ischemic attack; tPA: tissue plasminogen activator.

[a] OSA is defined as present when the AHI is ≥ 5 events/hour.

[b] p Values < 0.05

[c] Large neck is defined as > 16″ in women, > 17″ in men.

[d] Low ejection fraction is defined as an ejection fraction < 50%.

[e] Presence of PFO on echocardiogram during hospitalization or prior history of PFO.

Table 2

Stroke location and presence of obstructive sleep apnea.

Stroke Location	Overall (n=73)	OSA[a] (n=58)	No OSA (n=15)	*p*-value
Lobar	38 (52.1)	32 (55.2)	6 (40.0)	0.29
Frontal	19 (26.0)	15 (25.9)	4 (26.7)	0.99
Parietal	16 (21.9)	15 (25.9)	1 (6.7)	0.17
Temporal	9 (12.3)	6 (10.3)	3 (20.0)	0.38
Insular	9 (12.3)	8 (13.8)	1 (6.7)	0.67
Occipital	10 (13.7)	9 (15.2)	1 (6.7)	0.68
1 lobe involved	21 (28.8)	18 (31.0)	3 (20.0)	0.53
2 lobes involved	11 (15.1)	9 (15.2)	2 (13.3)	0.99
> 2 lobes involved	6 (8.2)	5 (8.6)	1 (6.7)	0.99
Subcortical	32 (43.8)	25 (43.1)	7 (46.7)	0.80
Thalamus	8 (11.0)	7 (12.1)	1 (6.7)	0.99
Basal ganglia	12 (16.4)	8 (13.8)	4 (26.7)	0.25
Internal capsule	10 (13.7)	10 (17.24)	0	0.11
Corona radiata	16 (21.9)	13 (22.4)	3 (20.0)	0.99
Corpus callosum	2 (2.7)	1 (1.7)	1 (6.7)	0.37
Brainstem	8 (11.0)	5 (8.6)	3 (20.0)	0.35
Midbrain	1 (1.4)	1 (1.7)	0	0.99
Pons	7 (9.6)	4 (6.9)	3 (20.0)	0.15
Medulla	1 (1.4)	1 (1.7)	0	0.99
Cerebellum/cerebellar peduncles	6 (8.2)	4 (6.9)	2 (13.3)	0.60
ACA territory	3 (4.11)	3 (5.2)	0	0.99
MCA territory	13 (17.8)	11 (19.0)	2 (13.3)	0.99
PCA territory	3 (4.11)	3 (5.2)	0	0.99

Shown are n (%). ACA: anterior cerebral artery; MCA: middle cerebral artery; OSA: obstructive sleep apnea; PCA: posterior cerebral artery.

[a]OSA is defined as present when the apnea-hypopnea index is ≥ 5 events/hour.

Table 3

Polysomnographic features among acute stroke patients.

Stroke Location	OSA Prevalence	AHI	OAI	CAI	Arousal Index[a]	Percent time SaO_2< 90%
Overall	58 (79.5)	12.2±13.9	4.6±10.4	1.1±4.2	20.9±11.7	3.9±8.6
Lobar	32 (84.2)	14.5±16.8	6.3±13.6	1.4±4.3	21.8±12.2	5.6±8.9
Frontal	15 (79.0)	15.0±17.3	5.3±8.9	2.0±5.6	21.6±11.6	9.2±11.4
Parietal	15 (93.8)	18.6±21.2	9.3±19.3	0.1±0.2	20.9±13.0	6.8±9.9
Temporal	6 (66.7)	9.3±11.0	6.3±8.6	1.8±4.9	26.3±8.7	3.0±5.1
Insular	8 (88.9)	11.9±12.3	2.2±4.2	1.1±3.1	14.8±5.0	8.9±13.6
Occipital	9 (90.0)	13.3±11.4	6.9±8.4	0.7±1.7	23.3±11.7	5.1±10.2
1 lobe involved	18 (85.7)	13.9±17.2	7.3±16.9	1.7±4.8	23.7±14.1	3.1±5.9
2 lobes involved	9 (81.8)	17.6±18.9	4.0±6.4	1.4±4.1	17.4±8.2	9.2±10.2
>2 lobes involved	5 (83.3)	10.5±10.7	7.6±10.2	0.1±0.2	23.1±9.3	8.0±14.5
Subcortical	25 (78.1)	9.1±9.3	2.3±4.0	1.4±5.0	23.3±11.5	4.6±10.0
Thalamus	7 (87.5)	7.7±8.8	3.3±4.2	1.8±4.9	24.1±9.3	2.6±5.4
Basal ganglia	8 (66.7)	8.7±8.8	1.3±1.5	2.3±7.4	19.2±10.0	5.0±13.5
Internal capsule	10 (100))	12.8±9.0	3.4±3.4	1.6±4.4	28.6±12.3	7.1±14.3
Corona radiata	13 (81.3)	12.2±10.2	2.5±4.6	1.7±6.1	23.5±13.7	3.7±7.8
Corpus callosum	1(50.0)	3.5±4.9	0.0±0.0	0.0±0.0	18.7±16.4	5.9±8.3
Brainstem	5 (62.5)	11.0±9.6	4.6±6.9	0.0±0.0	13.2±8.8	0.4±0.5
Midbrain	1 (100)	27.8	21.0	0.0	31.3	0.5
Pons	4 (57.1)	10.0±10.0	4.8±7.4	0.0±0.0	13.2±9.5	0.4±0.6
Medulla	1 (100)	17.7	3.0	0.0	12.9	0.6
Cerebellum/cerebellar peduncles	4 (66.7)	11.2±13.0	3.2±5.0	0.0±0.0	17.7±7.5	1.3±2.1
ACA territory	3 (100)	8.3±3.7	3.0±2.1	0.0±0.0	15.5±0.8	0.7±0.5
MCA territory	11 (84.6)	7.5±6.1	1.1±1.8	0.1±0.1	16.6±9.9	4.7±8.8
PCA territory	3 (100)	16.6±14.4	1.1±0.6	0.2±0.2	16.1±4.2	0.6±0.6

Shown are mean±SD or n (%). ACA: anterior cerebral artery; AHI: apnea hypopnea index; CAI: central apnea index; MCA: middle cerebral artery; OAI: obstructive apnea index; OSA: obstructive sleep apnea; PCA: posterior cerebral artery; SaO_2: oxygen saturation.

[a] Arousal index is the average number of arousals per hour (measure of sleep fragmentation)

Table 4

Multivariable modeling results.

Stroke Location	OSA Prevalence[a]				AHI[b]			
	Unadjusted		Adjusted[c]		Unadjusted		Adjusted[c]	
	OR (95% CI)	*p*- value	OR (95% CI)	*p*- value	Parameter Estimate (95% CI)	*p*- value	Parameter Estimate (95% CI)	*p*- value
Posterior circulation	0.97 (0.29, 3.25)	0.97	1.60 (0.40, 6.44)	0.51	0.10 (−0.39, 0.60)	0.68	0.17 (−0.30, 0.64)	0.48
Anterior circulation	1.73 (0.50, 5.97)	0.39	1.10 (0.26, 4.61)	0.89	0.20 (−0.34, 0.73)	0.48	0.22 (−0.31, 0.75)	0.41
Subcortical/lacunar	0.33 (0.08, 1.30)	0.11	0.52 (0.12, 2.24)	0.38	−0.34 (−0.82, 0.13)	0.16	−0.33 (−0.79, 0.13)	0.16
Supratentorial	2.14 (0.61, 7.52)	0.24	1.46 (0.34, 6.25)	0.61	0.18 (0.39, 0.75)	0.54	0.16 (−0.39, 0.72)	0.57
Infratentorial	0.37 (0.10, 1.33)	0.13	0.61 (0.14, 2.71)	0.51	−0.16 (−0.74, 0.43)	0.60	−0.14 (−0.71, 0.43)	0.64
Potential motor pathways	0.56 (0.14, 2.21)	0.40	0.79 (0.18, 3.49)	0.75	−0.23 (−0.74, 0.29)	0.39	−0.09 (−0.60, 0.42)	0.73

Shown are odds ratio (95% confidence interval) or parameter estimate (95% confidence interval). AHI: apnea-hypopnea index; CI: confidence interval; OR: odds ratio; OSA: obstructive sleep apnea.

[a] Logistic regression was used to model the outcome of OSA prevalence.

[b] Negative binomial regression was used to model the AHI.

[c] The following characteristics were used as covariates in the multivariable modeling: neck circumference and body mass index.